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VAGOTIMISED DOGS, WITH ESPECIAL REFERENCE
TO ATRIO-VENTRICULAR RHYTHM.

BY THOMAS LEWIS AND PAUL D. WHITE.

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THE EFFECTS OF PREMATURE CONTRACTIONS IN
VAGOTIMISED DOGS, WITH ESPECIAL REFERENCE TO ATRIO-
VENTRICULAR RHYTHM.

BY THOMAS LEWIS* AND PAUL D. WHITE.

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Introductory.

IN the present communication we discuss chiefly the effects of premature contractions upon atrio-ventricular rhythm as they have been observed in some recent experiments. We have used dogs exclusively and have made our observations in great part, upon animals which form the basis of another communication by one of us.⁶ We have worked upon animals fully anæsthetised with morphia, paraldehyde and ether, in which both vagi were severed in the neck. The chest wall being opened, artificial respiration was in progress. For the method of producing atrio-ventricular rhythm and for a description of the method of recording the heart beat, we may refer to the same paper. Premature contractions were induced from various points by means of single induction shocks, at or about threshold value, and thrown into the heart at suitable intervals. We found at an early stage of the experiments that very exact measurements are required of the intervals of the curves. We have taken our records upon photographic plates and have measured our intervals on these plates by means of the Lucas comparator. Of these measurements a number of examples are given in the accompanying tables. We have paid considerable attention to the rate and uniformity of travel of our plates and the accuracy of our rotatory timemarker, measuring all time marks adjacent to points on the electrocardiogram in each of our curves. The chief source of error is in fixing the cross wires of the microscope upon the deflections measured. Magnified, the beginning of auricular deflections are often blunt, and the point taken is subject to choice within certain limits. But as a general rule the auricular complex shows in its initial phase some acute angle, notch or point, upon which cross wires may be fixed with considerable accuracy. The beginnings of the auricular complexes, which form one of the bases of our tables, have been corrected from additional measurements of this kind, and in this manner a changing error has been avoided very largely.

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That is to say, although we may not be able to give the actual onset of an auricular complex with very great accuracy, the error in the interval between two complexes of the same kind is minute, for if there be a small error, it is maintained. In comparing successive intervals this method of mensuration has very obvious advantages. The error in fixing the onset of an auricular complex is small; we take the actual onset at a calculated time from a given notch or angle, basing our calculation upon average measurements of the actual onset in the curve. We have used those curves only which provide sharp points for measurements; consequently, every precaution has been taken to obtain electrocardiograms as smooth and uniform as possible. The measurement of excited contractions has called for additional care; in most of our curves there is no trace of escape from the stimulating electrodes, a fact which has been ascertained by a comparison of make and break responses, and by searching the curves where break excitations fall in refractory periods.* But in all cases we have checked our measurements by comparing the sharp movement of the excitation signal. The great majority of our measurements have been repeated; often by an independent observer.

Working in this fashion, we have reduced our errors of measurement to very small quantities, often the maximal error, as we calculate it from variations in timemarker, speed of plate travel and points chosen, amounts to one-thousandth of a second or less. The maximal error is certainly no greater in any case than four or five thousandths. The variation in successive and similar *P-R* intervals in any table gives a fair idea of the maximal error. In our tables we have consequently given our figures to four places of decimals, so as to render the third place as accurate as possible.

Our measurements have been taken from electrocardiograms because these curves are of far greater accuracy than myocardiograms. We have almost always taken simultaneous curves directly from the muscle of the right appendix and across the ventricles, but the upstrokes of mechanical curves have no sharpness, the error in measurements is in hundredths and not thousandths of a second. Moreover, as has become plain to us, the myocardiogram introduces another source of inaccuracy when its curves are taken to indicate the onset of contractions. The interval which elapses between the onset of contraction in a chamber and the arrival of this contraction at the region to which the mechanical device is attached is by no means negligible. The error may amount to as much as four or more hundredths of a second, varying according to the relative position of the levers and onset of contraction and the size of the heart. An illustrative curve is shown in Fig. 4, where a contraction excited from the right appendix interrupts an atrio-ventricular rhythm. The points of onset of the auricular excitation waves are clearly marked in the electrocardiograms; the *P-A*† distance for the atrio-ventricular beat is .0510 seconds, for the appendix

* Where the excitation shows it is clearly separate from the auricular complex

† Auricular complex to movement of auricular lever.

beat it is only .0073 seconds. Our experience has convinced us that the complex effects of premature contractions cannot be studied with accuracy unless studied electrocardiographically, for the errors introduced in mechanical tracings are of a sufficiently high order to vitiate the results.

Our mechanical curves have served nevertheless as useful checks to the correct interpretation of the electric events.

Definitions.

In discussing the effects of premature contractions upon the rhythms which they disturb, it will be necessary for us to refer repeatedly to individual contractions of auricle or ventricle and to individual intervals between contractions. For the sake both of brevity and clearness we shall employ a fixed and simple terminology. The beats and cycles which precede a disturbance and which belong to the dominant rhythm we shall term the "initial beats" and "initial cycles" (Fig. 1, I.C.). The premature contraction we shall term the "forced beat," and shall apply this term to auricular (Fig. 1, F.B.) and ventricular systole without regard to the chamber in which such beat arises. The interval between the last initial beat and the forced beat will be referred to as the "forced cycle" (F.C.A. and F.C.V.). The contractions which follow the disturbance will be named "restored beats," the intervals between them the "restored cycles" (Rs. C.A.). The interval between forced beat and the first restored beat will be termed the "returning cycle" (R.C.A. and R.C.V.). The forced cycle and returning cycle will be referred to together as the "conjoined cycles" (C.C.). An *As-Vs*, or *Vs-As*, interval, will be qualified by the adjective attached to the *cycle* which it terminates.

1. THE EFFECTS OF PREMATURE AURICULAR CONTRACTIONS UPON *S-A* RHYTHM.

Our observations have been chiefly upon *A-V* rhythm, but we considered it desirable to have parallel observations upon *S-A* rhythm* in the same animals. We shall report these observations briefly, emphasising the fact that we are dealing with very accurate measurements.

Our curves show the events which are generally recognised to accompany premature auricular contractions. We have applied our excitations in the main at three points, namely to right and left appendices, and over inferior vena cava; we have used the right appendix especially.

Changes in As-Vs intervals. The forced *As-Vs* interval varies with two factors. It varies according to the site at which the excitation is applied. It is longer when the right appendix is stimulated than when inferior cava is stimulated by some one or two hundredths of a second. These differences

* So far as we are aware no systematic observations have as yet been made electrocardiographically.

we naturally attribute to the different lengths of muscle traversed. They might be used as actual measurements of the rate at which the excitation wave travels, but in practice we do not find them to be capable of sufficiently exact expression. We find clear evidence that the forced *As-Vs* interval is longer the more premature the forced beat; a fact which is fully illustrated by our tables,* and which we believe has received no conclusive demonstration for the normally beating mammalian heart hitherto. The changes are small, being measured as a rule in one or two hundredths or in thousandths of a second. We find also invariable evidence of reduction of the returning *As-Vs* interval, unusually to be measured in hundredths (Fig. 2), usually in thousandths of a second; there is occasional though inconstant evidence that this interval is shortest when the returning cycle is longest. The shortening may be confined to the returning *As-Vs* interval, it may also affect the succeeding interval, though this is rare.

Lengths of returning and restored cycles in auricle. The length of the returning cycle appears to be governed by several factors, the most important of which is the rate at which impulses are elaborated at the pacemaker. A change of original sinus rate immediately changes the length of returning pause *and by an approximately equivalent amount*. We consider that this fact has not received due emphasis in the past, for it clearly shows that the actual length of pause is governed in the main by the rate of impulse formation in the *S-A* node. It has been stated by Cushny and Matthews¹ and also by Hering³ that the length of the returning pause is greater when the forced beat is more premature; but we fail to find clear evidence for this statement in their curves. We have looked for a more precise change, namely, a change in the length of the returning cycle, relative to the initial cycles, and for this we have conclusive data. The range is however small, being measured often in thousandths of a second or at the most one, two, three or four hundredths. For reasons which we consider later we are not convinced that Wenckebach's explanation, namely, difference in the rate of conduction to the pacemaker accounts for these differences.

In calculating the actual excess of the returning cycle over an initial cycle, we have been surprised at the smallness of these values; in stimulating right appendix or inferior vena cava they amount to no more than 3, 4 or 5 hundredths of a second as a general rule in medium sized dogs. On no occasion has the value exceeded the natural *As-Vs* interval for the same animal; as a rule it does not reach to half this amount. We have measured the distances over which the contraction wave would travel by direct spread from the point of excitation to the pacemaker, and have calculated the approximate time which such contractions waves would take in travelling, on the basis of recent measurements of the speed of travel (Lewis⁵). These data are not sufficiently abundant for conclusive deductions,† but they are

* Such few exceptions as are noted are covered by our error of measurement.

† We have recently investigated the point by more exact methods, and have been able to show a definite relation between excess of length of the returning cycle and the transmission time.

TABLE I. S.A. RHYTHM. STIMULATION OF AURICLE (SINGLE). 339

Dog.	Point of Stimulation.	Initial Cycles.		Forced Cycle.*	Returning Cycle.	Restored Cycles.			
		P-P	P-R			P-P	P-R	R-R	
EF. (1843)	Rt. app.	P-P	·7424	·7483	·5618	·7997	·7391	·7343	·7344
		P-R	·1046	·1079	·1040	·1181	·1044	·1038	·1044
		R-R	·7457	·7444	·5759	·7860	·7385	·7349	·7337
EF. (1843)	Rt. app.	P-P	·7900	·7916	·5787	·8549	·7903	·7936	·7927
		P-R	·1053	·1060	·1060	·1141	·1024	·1084	·1093
		R-R	·7907	·7916	·5868	·8432	·7963	·7945	·7894
EJ. (1945a)	Rt. app.	P-P	·5652	·5631	·4092	·6548	·5846	·5656	
		P-R	·1343	·1323	·1301	·1423	·1200	·1274	·1273
		R-R	·5632	·5609	·4214	·6325	·5920	·5655	
EJ. (1945b)	Rt. app.	P-P	·5656	·5580	·4961	·6163	·5625	·5583	
		P-R	·1274	·1273	·1345	·1435	·1208	·1304	·1321
		R-R	·5655	·5652	·5051	·5904	·5721	·5600	
EJ. (1970) Fig. 2	Inf. cav.	P-P	·6601	·6622	·4211	·7741	·6699	·6623	
		P-R	·1369	·1368	·1352	·1419	·0982	·1362	·1352
		R-R	·6600	·6606	·4278	·7304	·7097	·6613	
EJ. (1963)	Inf. cav.	P-P	·6614	·6617	·4617	·7690	·6714	·6616	·6635
		P-R	·1362	·1382	·1382	·1403	·1012	·1359	·1367
		R-R	·6634	·6617	·4638	·7299	·7061	·6624	·6632
EL. (2039)	Rt. app.	P-P	·3243	·3207	·1675	·3658	·3280	·3194	·3213
		P-R	·1039	·1034	·1051	·1525	·1047	·1047	·1040
		R-R	·3238	·3224	·2149	·3180	·3280	·3187	·3211
EL. (2037b)	Rt. app.	P-P	·3145	·1353	·2169	·3594	·3184	·3137	·3156
		P-R	·1051	·1042	·1047	·1282	·1047	·1063	·1055
		R-R	·3136	·3158	·2404	·3359	·3200	·3129	·3144
EL. (2037a)	Rt. app.	P-P	·3143	·3151	·2556	·3531	·3151	·3156	
		P-R	·1038	·1048	·1055	·1180	·1043	·1059	·1056
		R-R	·3153	·3158	·2681	·3394	·3167	·3153	
EL. (2050a)	Inf. cav.	P-P	·3675	·3670	·2866	·4164	·3683		
		P-R	·1069	·1076	·1073	·1040	·1046	·1064	
		R-R	·3682	·3667	·2833	·4170	·3701		
EL. (2048a)	Inf. cav.	P-P	·3600	·3601	·3031	·4039	·3578		
		P-R	·1089	·1082	·1081	·1031	·1039	·1093	
		R-R	·3593	·3600	·2981	·4047	·3632		
EL. (2048b)	Inf. cav.	P-P	·3621	·3590	·3230	·3959	·3606		
		P-R	·1093	·1069	·1094	·1028	·1044	·1079	
		R-R	·3597	·3615	·3164	·3975	·3641		
EL. (2050b) (Fig. 5)	Inf. cav.	P-P	·3663	·3690	·3340	·3998	·3661	·3661	
		P-R	·1064	·1078	·1056	·1038	·1038	·1071	·1088
		R-R	·3677	·3668	·3322	·3998	·3694	·3678	
EM. (2088a)	Rt. app.	P-P	·2989	·3019	·2000	·3534	·3047	·2993	
		P-R	·0989	·1022	·0981	·1302	·0995	·0981	·1015
		R-R	·3022	·2978	·2321	·3227	·3033	·3027	
EM. (2088b)	Rt. app.	P-P	·2993	·3000	·2266	·3382	·3043	·3016	
		P-R	·1006	·1003	·1000	·1175	·1006	·1010	·1006
		R-R	·2990	·2997	·2441	·3213	·3047	·3012	
EM. (2098b)	Rt. app.	P-P	·3028	·3052	·2608	·3293	·3043	·3043	·3027
		P-R	·1036	·1028	·1032	·1076	·1012	·1037	·1027
		R-R	·3020	·3056	·2652	·3229	·3068	·3033	·3033
EM. (2098a)	Rt. app.	P-P	·3018	·3035	·2776	·3239	·3044	·3038	·3030
		P-R	·1005	·1004	·1000	·1055	·1004	·1026	·1026
		R-R	·3017	·3031	·2831	·3188	·3066	·3038	·3020

* In this and subsequent tables the forced cycle is marked in heavy type in the auricular or ventricular line, according to the point of application of the stimulus.

TABLE II. S.A. RHYTHM. SINGLE AND SUCCESSIVE STIMULATION.

Dog.	Point of Stimulation.	Initial Cycles.		Forced Cycle.	Additional Forced Cycles.		Returning Cycle.	Restored Cycles.	
		P-P	R-R		P-P	R-R		P-P	R-R
EX. (2158)	Rt. app.	.3072	.3090	.1857			.3876	.3101	.3075
	P-R	.0822	.0816	.0800			.1347	.0802	.0814
	R-R	.3066	.3074	.2404			.3331	.3113	.3055
EX. (2159a)	Rt. app.	.3099	.3110	.2320			.3795	.3106	.3089
	P-R	.0808	.0799	.0798			.1260	.0772	.0796
	R-R	.3090	.3109	.2782			.3307	.3130	.3091
EX. (2159b)	Rt. app.	.3052	.3064	.2482	1.5793 (6 cycles)		.3614	.3088	.3075
	P-R	.0791	.0784	.0823	.1000	.0998	.1025	.0751	.0779
	R-R	.3045	.3013	.2659	.2609	1.5820 (6 cycles)	.3340	.3116	.3086
EZ. (2186a)	Rt. app.	.4122	.4124	.2400			.4833	.4305	.4188
	P-R	.0767	.0776	.0780			.1036	.0798	.0784
	R-R	.4131	.4128	.2656			.4586	.4300	.4183
EZ. (2186c)	Rt. app.	.4221	.4234	.2351	.2545	.2540	.4802	.4474	.4277
	P-R	.0781	.0767	.0764	.1024	.1028	.1027	.0775	.0685
	R-R	.4207	.4231	.2611	.2549	.2539	.4550	.4384	.4265
EZ. (2186b)	Rt. app.	.4214	.4223	.2072	1.3951 (5 cycles)		.4901	.4383	.4278
	P-R	.0774	.0782	.0787	.1036		.1009	.0785	.0780
	R-R	.4222	.4228	.2321	1.3924 (5 cycles)		.4677	.4378	.4263
EZ. (2187b)	Rt. app.	.4703	.4719	.3086	.8806 (3 cycles)		.5189	.4868	.4754
	P-R	.0899	.0900	.0892	.1021		.1035	.0893	.0875
	R-R	.4704	.4711	.3215	.8820 (3 cycles)		.5047	.4850	.4747
EZ. (2187a)	Rt. app.	.4699	.4681	.3502	1.3323 (4 cycles)		.5206	.4761	.4759
	P-R	.0905	.0905	.0907	.1021		.1022	.0864	.0910
	R-R	.4699	.4683	.3616	1.3324 (4 cycles)		.5048	.4807	.4729
EZ. (2187c)	Rt. app.	.4754	.4731	.4027	1.5002 (5 cycles)		.5395	.4978	.4887
	P-R	.0902	.0895	.0918	.1000		.1080	.0793	.0833
	R-R	.4747	.4754	.4109	1.5082 (5 cycles)		.5108	.5018	.4860

not incompatible with Wenckebach's view that the increased length of the returning pause is due in the main at least to the time lost while the contraction wave is travelling to the pacemaker.

For example in Table I, Curves 2037*a* and 2050*b*, the excess of the returning pause in the auricle over the initial pause is .0384 seconds for right appendix stimulation, and .0322 for inferior caval stimulation. The distance of these points from the upper end of the sulcus terminalis was, with the auricle in the diastolic position, approximately the same, namely, 30 mm.. In four animals, one of us found the average rate of conduction in the auricle to be 955 mm. per second; the time taken to travel 30 mm. would be .031 seconds, calculated upon this basis.

The first restored cycle is almost always longer than the initial cycles, but by trifling amounts (usually 3, 4 or 5 thousandths of a second, on one occasion it reached one hundredth, on another two hundredths of a second). It is possible, as Cushny² has suggested, that a retardation of the *S-A* impulse formation, produced by the premature beat may play some part in prolonging the returning cycle, but we think that evidence for this view is still lacking. It certainly appears to play little part in the vagotomised animal. As Table II shows the excess of the returning over initial cycles may be less after successive than after single interruptions; although there may be more lengthening of the restored cycles in the first named circumstance.

The conjoined cycles are together shorter than two initial cycles in all our curves, with a few solitary exceptions to be subsequently considered; they are shortest when the forced beat is most premature. The exceptions are instances where the contraction wave has evidently failed to reach the pacemaker. Of such curves we shall speak again.

2. THE EFFECT OF PREMATURE AURICULAR CONTRACTIONS UPON *A-V* RHYTHM.

Hering⁴ and Rothberger and Winterberg⁷ have already made some observations upon the effects of auricular extrasystoles upon *A-V* rhythm. These writers describe two forms of disturbance, one of which is limited to the auricle, the other affecting both chambers. In the former instance the ventricle is said to suffer no disturbance; in the last instance it is stated that despite conduction to the lower chamber the returning pause may be compensatory. Hering used myocardiographic curves only, Rothberger and Winterberg employed electrocardiograms in addition. In both series of observations, attention appears to have been focussed upon the question of compensation, individual pauses have received no detailed analysis.

We may introduce the chief events by means of a figure (Fig. 3). In this figure the initial beats are of *A-V* nodal origin, the systoles of auricle and ventricle being almost simultaneous; the interval is .037 seconds. The premature contraction is excited from the inferior cava, and the *forced auricular beat* (F.B.) is followed by a *forced ventricular beat*. The lengths

of the forced cycles in auricle and ventricle are very different, and for the reason that the relations of corresponding auricular and ventricular systole are different. The forced cycle in the auricle is shorter than in the ventricle by six hundredths of a second; it is shorter by the forced *As-Vs* interval less the last of the initial *As-Vs* intervals. Conversely, the *returning cycle* in the auricle (R.C.A.) is longer than that in the ventricle (R.C.V.); it is longer by the forced *As-Vs* interval less the returning *As-Vs* interval.

Forced cycle in auricle. It will be clear that the position of the forced beat in the auricle is controlled absolutely by the excitation; it may commence at any period of the auricular diastole. Its position controls the length of the forced auricular cycle.

Forced cycle in ventricle. The position of the forced ventricular beat is controlled by the time at which excitation occurs and by the interval which elapses between the excitation and the final response of the ventricle. This interval is composed for the most part of the forced *As-Vs* interval. The latency of the forced auricular contraction to stimulation we find to be very constant in a given animal under the conditions of our experiments.

The length of the forced ventricular cycle depends therefore upon:—

1. The position of the last initial ventricular beat.
2. The time of excitation in relation to this beat.
3. The length of the forced *As-Vs* interval.

Forced As-Vs interval. We find this interval to vary appreciably in different circumstances in the same animal. As might be expected from similar changes in *S-A* rhythm, it is of greater length when the forced beat comes early and of shorter length when this beat comes late in diastole. The actual variation in length is definite, as the measurements given in our tables show, it amounts to a few thousandths of a second in most experiments, at the most to a few hundredths (Table III). Another factor which influences the length of this interval is the position at which stimulation is applied to the auricle. Evidently the time taken for contraction to travel from auricle to ventricle must vary according to the point from which the contraction starts; it is chiefly a question of the degree of variation. We find that the differences are measurable though they cannot be very accurately expressed. Thus, in Table III (Dog EL.) the *As-Vs* interval for right appendicular stimulation is approximately .01 seconds longer than for inferior caval stimulation; compared with the normal *As-Vs* interval in the same animal, the inferior caval *As-Vs* interval is reduced (see Table I, Dog EL.) while the right appendicular *As-Vs* interval is a little increased.

TABLE III. A.V. RHYTHM. STIMULATION OF AURICLE (SINGLE). 343

Dog.	Point of Stimulation.		Initial Cycle.	Initial Cycle.	Forced Cycle.	Returning Cycle.	Restored Cycle.		
EE. (1822a)	Rt. app.	P-P		·6739	·4673	·7756	·6709		
		P-R	·0565	·0589	·0607	·1169	·0606	·0606	
		R-R	·6764	·6757	·5235	·7193	·6709		
EE. (1813)	Rt. app.	P-P	·6910	·6912	·5119	·7908	·6894	·6876	·6884
		P-R	·0540	·0562	·0551	·1122	·0541	·0550	·0547
		R-R	·6932	·6901	·5690	·7327	·6903	·6873	·6895
EE. (1822b)	Rt. app.	P-P	·6705	·6724	·6094	·7308	·6721		
		P-R	·0606	·0619	·0596	·1106	·0589	·0603	
		R-R	·6718	·6701	·6604	·6791	·6735		
EF. (1850)	Rt. app.	P-P	·8397	·8368	·2869	1·1174	·8498	·8431	
		P-R	—·0555	—·0575	—·0557	+·1155	—·0526	—·0540	—·0522
		R-R	·8377	·8386	·4581	·9493	·8484	·8449	
EF. (1838)	Rt. app.	P-P	·8254	·8284	·3165	1·0858	·8498	·8326	
		P-R	—·0519	—·0471	—·0463	+·0975	—·0521	—·0517	—·0475
		R-R	·8302	·8292	·4603	·9362	·8502	·8368	
EJ. (1972)	Rt. app.	P-P	·6797	·6814	·3808	·7754	·6887	·6905	
		P-R	·0880*	·0877	·0875	·1515	·0916	·0920	·0880
		R-R	·6794	·6812	·4448	·7155	·6891	·6865	
EJ. (1981)	Rt. app.	P-P	·6864	·6880	·4320	·7767	·6903	·6908	
		P-R	·0833	·0834	·0838	·1506	·0944	·0934	·0902
		R-R	·6865	·6884	·4988	·7205	·6893	·6876	
EJ. (1975)	Rt. app.	P-P	·6893	·6868	·4811	·7788	·6928	·6856	
		P-R	·0786	·0798	·0797	·1492	·0894	·0898	·0876
		R-R	·6905	·6867	·5506	·7190	·6932	·6834	
EJ. (1967) (Fig. 21)	Inf. cav.	P-P	·7170	·7227	·4301	·7612	·7143	·7180	·7017
		P-R	·0961	·0855	·0471	·1395	·0958	·0886	·0568
		R-R	·7064	·6843	·5225	·7175	·7071	·6862	·6955
EL (2041)	Rt. app.	P-P	·4480	·4461	·2432	·5165	·4509	·4496	
		P-R	·0335	·0334	·0328	·1129	·0417	·0378	·0334
		R-R	·4479	·4455	·3233	·4453	·4470	·4452	
EL. (2044) (Fig. 4)	Rt. app.	P-P	·4557	·4567	·3108	·5335	·4570	·4558	·4557
		P-R	·0258	·0254	·0258	·1095	·0324	·0297	·0268
		R-R	·4553	·4571	·3945	·4564	·4543	·4529	·4551
EL. (2043)	Rt. app.	P-P	·4477	·4476	·3675	·5139	·4489	·4473	·4486
		P-R	·0234	·0241	·0262	·1078	·0275	·0260	·0274
		R-R	·4484	·4497	·4491	·4336	·4474	·4487	·4448
EL. (2053)	Inf. cav.	P-P	·5572	·5563	·2349	·6320	·5686		
		P-R	·0397	·0398	·0385	·1099	·0431	·0366	
		R-R	·5573	·5550	·3063	·5652	·5621		
EL. (2054)	Inf. cav.	P-P	·5492	·5453	·2694	·6178	·5630	·5470	
		P-R	·0352	·0350	·0354	·1026	·0442	·0371	·0372
		R-R	·5490	·5457	·3366	·5594	·5559	·5471	
EL. (2052)	Inf. cav.	P-P	·5469	·5459	·3079	·6144	·5556	·5479	
		P-R	·0337	·0340	·0371	·0975	·0407	·0378	·0354
		R-R	·5472	·5490	·3683	·5576	·5527	·5455	
EL. (2051)	Inf. cav.	P-P	·5411	·5399	·3289	·6126	·5536	·5422	
		P-R	·0367	·0376	·0394	·0984	·0413	·0387	·0379
		R-R	·5420	·5417	·3879	·5555	·5510	·5414	
EM. (2085)	Rt. app.	P-P	·4294	·4308	·2870	·5124	·4323	·4289	·4290
		P-R	·0221	·0223	·0204	·1000	·0222	·0225	·0214
		R-R	·4296	·4289	·3666	·4346	·4326	·4278	·4301
EM. (2084)	Rt. app.	P-P	·4344	·4295	·3413	·5004	·4312	·4286	
		P-R	0236	·0212	·0242	·1033	·0236	·0228	·0237
		R-R	·4320	·4325	·4204	·4207	·4304	·4295	

* The natural interval in this dog while A-V nodal rhythm was present was approximately ·0400 seconds. In these curves higher values are seen because the stimulation was frequent and each premature beat was followed by increased intervals for several cycles (see Fig. 21).

The returning auricular cycle. This cycle (R.C.A.) is the longest of the disturbance. Compared with the corresponding cycle in the ventricle (R.C.V.) it is longer by the difference of the forced and returning *As-Vs* intervals. Its length relative to the corresponding ventricular cycle may be said to depend absolutely upon the lengths of these two intervals. Consequently, if we consider the factors influencing the length of the returning cycle in the ventricle it will suffice. But it may be well to emphasise at this stage the complexity of the factors which determine the duration of the returning auricular cycle; it cannot be compared to the similar cycle which follows a disturbance of the *S-A* rhythm nor can it be taken as a measure of impulse formation in the *A-V* node.

The returning ventricular cycle. This cycle is the most important and our discussion will centre around it, for in length it evidently gives with most accuracy the time which elapses between the disturbance of the *A-V* node and the completion of the next *A-V* impulse. This inter-ventricular interval represents the corresponding inter-nodal interval, providing that conduction is at the same rate from node to ventricle for the two beats which bound it. Such divergence as might occur between inter-nodal and inter-ventricular intervals would be in the direction of shortening of the latter, for the forced node to ventricular interval should be longer on account of its prematurity. The length of the ventricular returning cycle may be compared with that of initial ventricular cycles. We find that, relative to the last named, its length is variable within certain limits; in the great majority of experiments it is prolonged (Table III), in exceptional cases it is sometimes prolonged and sometimes reduced. Where there is lengthening, this lengthening may amount to a few thousandths, or three or four hundredths of a second. In a solitary animal it amounted exceptionally to a tenth of a second. Where there has been a reduction of length, this reduction has amounted to a few thousandths of a second. Thus, the degree of lengthening or reduction, relative to the length of the initial cycle, is small. We may sum up in the statement that the returning cycle in the ventricle has approximately the same length as an initial cycle, but is usually subject to slight prolongation or exceptionally to slight reduction.

The actual length of the cycle is controlled by a single important factor, the rate of impulse formation in the *A-V* node. If the length of the cycle varies materially from time to time, a similar variation will be found in the initial cycles, clearly showing that the length depends upon the returning *A-V* automaticity.

The length of the returning ventricular cycle is not constantly influenced by the prematurity of the forced beat; but it is the rule that the more premature the beat the longer the returning pause. The variations are usually in thousandths of a second. The fact that conspicuous prematurity of the forced beat does not shorten the returning pause, as might be expected, but is usually accompanied by slight lengthening, leads us to the belief that the greater part of the ordinary increase in the forced *As-Vs* interval of conspicuously

premature beats occurs in the highest reaches of the node, or above the point at which the *A-V* rhythm originates; for if it occurred in the lower reaches of the junctional tissues, it would surely shorten the pause in question. The observation that the returning pause in *S-A* rhythm and *A-V* rhythm is similarly affected by the degree of prematurity of the forced beat, is opposed to Wenckebach's view in respect of interruptions of the first rhythm. For if his explanation, that the difference is due to variations in conduction time from point stimulated to *S-A* node, were applied to *A-V* rhythm, then increased prematurity of a forced beat interrupting the last-named rhythm, should have no effect upon the returning pause or should shorten it.

The length of the returning cycle is uninfluenced by the site of stimulation. This is as might be anticipated, for variations of the time relations due to this cause are evidently in the transmission from excitation point to *A-V* node and not in the transmission time on the ventricular side of the *A-V* node. There are other factors which might possibly affect the returning cycle, but these will be discussed more conveniently at a later stage.

The returning As-Vs interval. This interval is subject to considerable change. It is the rule to find, not shortening as in interruptions of the *S-A* rhythm, but noticeable lengthening. This fact is clearly illustrated by our tables and is very distinct in Fig. 21.

The lengthening may amount to four or more thousandths of a second and sometimes to several hundredths of a second (Fig. 21) and may be spread over a number of cycles. In such circumstances and if the premature beats occur frequently the natural *As-Vs* interval may be concealed. Thus in Fig. 21 the natural interval for an *A-V* beat was .040 seconds; but frequent extrasystoles kept it prolonged to values of .09, .08 and .07 seconds (see Dog E.J., Table III). In some instances there is practically no change; in some animals there is reduction. It is a very noticeable fact that the instances of no change or of reduction occur in animals in which the original *As-Vs* interval (of *A-V* rhythm) is relatively long.

The usual lengthening of this interval is, so we consider, the result of *bettered* conduction through those portions of the junctional tissues which unite auricle and the seat of impulse formation. One of us has shown that vagal stimulation, which produces forward heart-block in *S-A* rhythm, produces a shortened *As-Vs* interval during nodal rhythm, and has argued that this change is the result of hindrance to conduction in the tissue between auricle and impulse centre. The lengthening of *As-Vs* interval after the returning cycle, we regard as the reverse of this phenomenon. Whereas, during *S-A* rhythm, the returning *As-Vs* interval is shortened, and this unquestionably as a result of *bettered* conduction, in *A-V* rhythm the interval is longer than usual, though, as we think, from the same primary cause. And support is lent to this view, since, when the *A-V* rhythm arises at low levels (witnessed by short initial intervals) the lengthening of the returning *As-Vs* interval is conspicuous. We cannot sufficiently emphasise the constancy

of our findings in one respect, and we have abundant material; the direction in which this *As-Vs* interval changes is dependent upon its original length. If the interval is originally long and indicative of a high origin of the impulse, the change is in same direction as when *S-A* rhythm prevails; if short and indicative of a low origin of the impulse, the change is always in the reverse direction. In the first instance the change is in tissue below, in the last, it is in tissue above the focus which starts the contraction. The difference is due not to differences in the site of changed conduction, but to the position of the automatic centre relative to this site. We have some fortunate observations from a single animal, which are exemplified in Table IX (Curves 2161*b* and 2169). In this experiment the level of the *A-V* rhythm altered during the course of the observations; and we were able to obtain two series of curves from the same animal, showing the changes in the returning *As-Vs* interval under the two conditions. When the original interval was long, the returning interval showed shortening; when the original interval was short, the returning interval showed lengthening.

The restored cycles. The first restored cycle generally shows slight lengthening as compared to the initial cycles. Exceptions have been witnessed in a few animals (Table III, Dog EE. and Table IX, Dog EX. and FA.). The lengthening and the shortening when it occurs, is of trifling amount, namely a few thousandths of a second. The lengthening is not maintained, the succeeding cycle fails to show it except in rare instances.

The conjoined cycles. The length of forced and returning cycle together, or as we term them, the conjoined cycles, is not materially different in auricle and ventricle, though usually somewhat longer in the ventricle. This relative lengthening in the ventricle is evidently due to the customary increase of the returning *As-Vs* interval. If the returning interval is decreased, the conjoined cycles are longer in the auricle. In none of our experiments have we discovered the conjoined cycles to be equal in length to two initial cycles, except under the special circumstances where no forced ventricular beat occurs, a condition hereafter considered. Evidently since the returning cycle remains of almost constant length, the conjoined cycles vary in duration mainly according to the length of the forced cycle. The returning cycle may, it is true, almost compensate for the short forced cycle when the forced beat is late in diastole, even though the latter finds response in the ventricle. Thus in Table III, Curve 1822*b*, the figure for the conjoined cycles in the ventricle is 1.3395, while the two initial cycles total 1.3419 seconds. Here the difference is but two thousandths of a second, and it is evident that had the forced beat been still less premature the figures might have been equalised. But we regard the term compensatory, applied to any such phenomenon (Hering, Rothberger and Winterberg), as unfortunate,* since such restoration of the original

* The compensation witnessed in these experiments may possibly be attributed to the non-section of the vagi.

rhythm, if it occurs, comes about in a totally different manner to that described by Engelmann, in observing the effect of ventricular extrasystoles upon *S-A* rhythm. It would not be due to the control of the length of the returning cycle by a dominant rhythm, but to a balance between its lengthening and the shortening of the forced cycle; the former being almost constant in a given animal, the balance is clearly a matter largely of coincidence. The possible restoration of the original rhythm in this manner, if it occurs, should not deflect attention from the essential question, the length of the returning cycle as opposed to that of the conjoined cycles.

Of compensatory pauses.

During S-A rhythm. As we have already stated, amongst our curves, we find no instances in which the conjoined cycles are equal to two initial cycles, with a few solitary exceptions. These are all disturbances in which the forced beat has fallen late in diastole. We publish two of our examples. The first is seen in Fig. 6. Here the forced auricular cycle (as measured

TABLE V.

(COMPENSATORY BEATS). *S-A* RHYTHM. INFERIOR CAVAL STIMULATION.

Dog.		Initial Cycles.		Forced Cycle.	Returning Cycle.	Restored Cycle.		
EL. (2048) (Fig. 6)	<i>P-P</i>	·3606	·3601	· 3514	·3744	·3600		<i>I.V.C.</i> auricle pre-
	<i>P-R</i>	·1044	·1079	·1083	·1073	·0167	·1075	cedes <i>S-A</i> auricle
	<i>R-R</i>	·3641	·3605	·3504	·3738	·3608		by ·009 seconds.
EL. (2047) (Fig. 7)	<i>P-P</i>	·3673	·3679	· 3679	·3667	·3685	·3707	<i>S-A</i> auricle precedes
	<i>P-R</i>	·1044	·1065	·1054	·1045	·1083	·1077	<i>I.V.C.</i> auricle by
	<i>R-R</i>	·3694	·3668	·3670	·3705	·3679	·3675	·0003 seconds.

TABLE VI.

(COMPENSATORY BEATS). *A-V* RHYTHM. STIMULATION OF RIGHT APPENDIX.

Dog.		Initial Cycles.		Forced Cycle.	Returning Cycle.	Restored Cycles.				
EL. (2040) (Fig. 8)	<i>P-P</i>	·4509	·4524	· 4055	·4895	·4477	·4493			
	<i>P-R</i>	·0342	·0344	·0327	·0778	·0323	·0360	·0344		
	<i>R-R</i>	·4511	·4507	·4506	·4440	·4514	·4477			
EM. (2087)	<i>P-P</i>	·4183	·4183	· 3670	·4602	·4188	·4176			
	<i>P-R</i>	·0234	·0207	·0204	·0700	·0230	·0212	·0203		
	<i>R-R</i>	·4156	·4180	·4166	·4132	·4170	·4167			
EX. (2161c)	<i>P-P</i>	·4130	·4133	· 3531	·4733	·4140	·4117			
	<i>P-R</i>	·0355	·0378	·0372	·0999	·0380	·0387			
	<i>R-R</i>	·4153	·4127	·4158	·4114	·4147				
EX. (2165)	<i>P-P</i>	·3679	·3687	· 3163	·4217	·3685	·3682	·3697	·3687	·3687
	<i>P-R</i>	·0361	·0392	·0424	·0944	·0386	·0422	·0419	·0433	·0454
	<i>R-R</i>	·3710	·3719	·3683	·3659	·3721	·3679	·3711	·3708	·3698
FB. (2206a)	<i>P-P</i>	·4265	·4271	· 3668	·4772	·4259	·4265	·4256	·4263	
	<i>P-R</i>	·0375	·0369	·0370	·0922	·0380	·0366	·0364	·0364	·0358
	<i>R-R</i>	·4259	·4272	·4220	·4230	·4245	·4263	·4256	·4257	

from excitation signal and actual onset of *P*) is .3514 seconds; the returning auricular cycle is .3744 seconds. The initial cycles average .3603 seconds. The major events are clear upon close examination of the curve. The auricular summit *P* of the forced beat is of unusual form; it is transitional between the normal *P* for this animal and the *P* excited from the inferior cava (see Fig. 5). The forced beat which occurs late in diastole has fallen one hundredth of a second earlier than the point at which the natural beat is expected; we have seen that in this animal the probable transmission time from inferior cava to pacemaker is three hundredths of a second (page 341); insufficient time has elapsed for it to anticipate the natural beat at the pacemaker and consequently two waves of contraction have been propagated and have met in the auricular walls; hence the shape of the electric complex; and hence the compensation, for impulse formation in the *S-A* node has remained undisturbed. We say undisturbed, but as a matter of fact it is very slightly delayed, for there is in reality a little overcompensation. To this overcompensation we shall refer again, trifling though it be. It is of interest to observe that although the forced beat in question failed to affect the sino-auricular node, it has affected the ventricle, for the corresponding ventricular beat is premature also.

For comparison with this curve we give another from the same animal (Fig. 7). Here again we are dealing with a beat forced from the inferior cava, but (again as calculated from the excitation signal) it starts at almost exactly the same instant as the expected natural beat. That is to say, it starts a little later than the forced beat of the last figure; *the corresponding electric curve consequently resembles the natural P more closely*, for the forced contraction* wave has had less start over the pacemaker contraction wave. Like the forced beat of Fig. 6 it has failed to reach the pacemaker, but as opposed to the last, it has also failed to affect the ventricle, whose beat falls at the natural time.† It is to be noted that the succeeding ventricular cycle is prolonged; true, the increase is within our maximal error of measurement, but in such disturbances the difference is always in the direction of lengthening. We are content at present to record the fact.

During A-V rhythm. A comparison of the curve which we now give (Fig. 8) with those of such figures as Fig. 3 and 4 should make it clear why, when premature auricular contractions disturb an *A-V* rhythm, there is no great range in the lengths of the forced ventricular cycles. Conduction being in the forward direction throughout for such forced beats, the forced ventricular cycle can never be so short as the forced auricular cycle. There is a difference in the range of lengths of the corresponding cycles for another reason; a forced auricular beat may occur, as Rothberger and Winterberg

* More correctly excitation wave, but it is convenient to speak of events in terms of contraction.

† Judged by *P-R* intervals in other curves from the same animal, transmission to the *A-V* node should be more rapid when the wave starts at the inferior cava than when it starts at the *S-A* node. We are consequently unable fully to account for the events of this curve.

have stated, so late in diastole that when it reaches the *A-V* tissues it finds them refractory. In these circumstances the returning ventricular cycle should be compensatory. We have not found it exactly so. In Fig. 8 it is less than compensatory by seven thousandths of a second, and this value is well outside our maximal error for this animal. The restored auricular cycles also show shortening, though in less degree. A precisely similar shortening of compensation is seen in each of our tabulated curves (Table VI).

We may not be able satisfactorily to account for this constant change, any more than we may be able to account for the reverse change where forced auricular beats fail to reach the sino-auricular node, but they serve definitely to show that although a forced contraction spreads over a limited area of the heart and does not actually reach the point from which impulses are generated, yet these forced beats may affect impulse formation in the dominating centre. We shall have occasion to note further examples of this phenomenon.

The origin of restored beats.

When *A-V* rhythm is disturbed by forced contractions, the restored beats show certain changes which we have noted already. The most noticeable event is a lengthening of the first *As-Vs* interval. This lengthening has been discussed and we regard it as the result of simple conduction change. This view is based largely upon the gradual change from longer interval to the original interval which occurs in some animals (see Fig. 21). From time to time and especially when the cooling of the *S-A* node is not great, another change is witnessed. It consists in the escape of the *S-A* node. An example is shown in Fig. 10. The first forced beat is followed by such an escape, after an interval of 1.0735 seconds, and this despite the length of the initial cycle which is 1.2690 seconds. Curiously the second forced beat of the same figure is followed by an atrio-ventricular contraction, although the returning cycle measures 1.3562 seconds. It is evident that the rate of impulse formation in the nodes has not remained constant, but that it has been disturbed by the occurrence of the forced beats. The more premature beat has quickened the *S-A* rhythm sufficiently to prevent its escape; yet this quickening is but temporary, for immediately afterwards an *A-V* beat is preceded by a pause of 1.3415 seconds. We have seen numerous examples of similar changes in the rates of impulse formation and, as illustrated, they may follow single beats forced from auricle or ventricle. And the escape may not be confined to a single cycle, it may occur as in Fig. 9 and 14 for several successive cycles; ultimately the *A-V* rhythm is always restored. The escape, too, is not confined to the *S-A* node, it may be from other centres which we are unable accurately to locate. The reason of such escapes we shall consider more fully in the sequel.

3. THE EFFECTS OF PREMATURE VENTRICULAR CONTRACTIONS UPON A-V RHYTHM.

Hering and Rothberger and Winterberg have already recorded certain of these effects; we shall describe the disturbances in more detail. In these experiments we have adopted a single point of ventricular stimulation, namely, the infundibulum of the right ventricle.

In A-V rhythm the disturbances which happen in the heart when the ventricle is excited (Fig. 11 to 14) may be said to be similar to those which occur when the stimulation is auricular, if we may regard the heart beating in this fashion as consisting of two similar chambers *A* and *V*, lying on opposite sides of the A-V node, but with similar relations to it. At all events this statement is true for the major changes. *The forced cycle in the ventricle* is controlled in its length by the position of the forced beat, and this is governed by the moment of excitation. In most instances the forced ventricular beat creates an auricular response, just as the forced auricular beat creates a ventricular response. The length of the *forced cycle* in the auricle is fixed by the moment of excitation in relation to the last auricular beat and by the length of the forced *Vs-As* interval.

TABLE VII.

A-V RHYTHM. STIMULATION OF RIGHT VENTRICLE (SINGLE).

Dog.		Initial Cycles.		Forced Cycle.	Returning Cycle.	Restored Cycles.			
EC. (1748)	P-P	.4704	.4732	.4371	.4782	.4771	.4720	.4722	.4727
	P-R	+ .0427	+ .0417	+ .0401	— .0822	+ .0373	+ .0378	+ .0406	+ .0408
	R-R	.4694	.4716	.3148	.5977	.4776	.4748	.4724	.4734
ED (1778)	P-P	.7591	.7593	.6133	.7791	.7693	.7601		
	P-R	+ .0355	+ .0338	+ .0354	— .1588	+ .0327	+ .0352	+ .0351	
	R-R	.7574	.7609	.4191	.9706	.7718	.7600		
ED. (1778b)	P-P	.7601	.7549	.6555	.7690	.7609	.7665	.7452	
	P-R	+ .0352	+ .0351	+ .0343	— .1571	+ .0315	+ .0347	+ .0330	+ .0354
	R-R	.7600	.7541	.4641	.9576	.7641	.7648	.7476	
EE. (1805)	P-P	.6518	.6517	.5405	.6909	.6431	.6515	.6498	
	P-R	+ .0591	+ .0549	+ .0594	— .0958	+ .0507	+ .0572	+ .0599	+ .0568
	R-R	.6476	.6562	.3853	.8374	.6496	.6542	.6467	
EE. (1791a)	P-P	.6651	.6667	.5549	.7039	.6636	.6620		
	P-R	+ .0564	+ .0596	+ .0547	— .0967	+ .0536	+ .0548	+ .0555	
	R-R	.6683	.6618	.4035	.8542	.6648	.6627		
EE. (1791b) (Fig. 12)	P-P	.6636	.6620	.6389	.6674	.6664	.6626	.6656	
	P-R	+ .0536	+ .0548	+ .0555	— .0929	+ .0552	+ .0570	+ .0597	+ .0590
	R-R	.6648	.6627	.4905	.8255	.6682	.6653	.6649	
EZ. (2175)	P-P	.5925	.5904	.5676	.6084	.5942	.5939	.5902	.5908
	P-R	+ .0328	+ .0329	+ .0331	— .0809	+ .0328	+ .0330	+ .0324	+ .0324
	R-R	.5926	.5906	.4536	.7221	.5944	.5933	.5902	.5908

The forced Vs-As interval. In the case of auricular extrasystoles, we are able to give very accurate measurements; in the case of ventricular

extrasystoles we speak with more reserve in regard to the lengths of certain intervals and cycles. For the forced auricular contraction is represented by an electric complex which is buried in the electrocardiogram of the forced ventricular beat. It is not always possible to identify this auricular complex; it is but occasionally that its onset can be estimated accurately. The electric complex of the auricle, responding on the one hand to a nodal impulse, on the other to a retrograde impulse from the ventricle, appears from a selection of our curves to be of constant form. In some instances, therefore, we are able to choose a notch or peak upon the retrograde complex and identify it with a similar notch or peak upon the nodal auricular complex (Fig. 12 and 14). In doing so we are guided to some extent by the intra-auricular distance as measured in the mechanically written curve. Each series of curves has to be regarded upon its intrinsic merits and while we are of opinion that the tabulated measurements which we publish are as accurate expressions as may be of the occurrences, we are somewhat more diffident in the conclusions which we draw from them, because of the difficulty in recognising the onset of the forced auricular beat with certainty. The main events, however, are perfectly clear.

In these experiments we find no conclusive evidence that forced $Vs-As$ intervals are longer than forced $As-Vs$ intervals. We have seen that the lengths of forced $As-Vs$ intervals vary according to the site of stimulation of the auricle; we may assume a similar variation in $Vs-As$ intervals forced from various ventricular points. The duration of forward or backward conduction cannot be satisfactorily compared by our present methods; especially is this true if the measurements are undertaken in mechanical curves; for the $As-Vs$ or $Vs-As$ interval, at the case may be, expresses not only the conduction interval in one or other direction but the distance of the point stimulated from the conducting tissues, and also, in the case of mechanical curves, the distance of these tissues and the point of excitation from the recording levers. We are convinced that these factors are by no means immaterial. We are also unable to arrive at any final conclusion as to the effect of prematurity of the forced beat upon the length of the forced $Vs-As$ interval. We do not consider our measurements to be sufficiently reliable in this respect.

The returning ventricular cycle. This cycle is the longest of the disturbance. Compared with the returning auricular cycle it is longer by the sum of the forced $Vs-As$ and the returning $As-Vs$ interval. Its length is controlled by these intervals and by the same factors which influence the returning auricular cycle, which we may proceed to discuss.

The returning auricular cycle. The length of this cycle evidently most closely corresponds to the interval elapsing between the disturbance of the $A-V$ node and the completion of the next $A-V$ impulse. It represents this internodal interval accurately, if conduction is at the same rate from node

to auricle for the two beats which bound it. And, as in the case of forced auricular beats, such divergence as might be expected would be in the direction of shortening of the inter-auricular cycle; for the forced *Vs-As* interval should be relatively longer, in virtue of its prematurity. As a matter of fact, the inter-auricular cycle has been longer than the initial cycle by a few hundredths or thousandths of a second in all but one instance (Table X, Curve 2196).

The returning As-Vs interval. We have been able to detect no constant change in the length of this interval as compared with the initial intervals; there is more often shortening than lengthening.

The restored cycles. The first restored cycle is usually lengthened by a few thousandths of a second, but sometimes it is shortened.

The *conjoined cycles* in the auricle have never been equal to two initial cycles, except when the forced ventricular contraction has failed to reach the auricle. The returning auricular cycle may be almost compensatory, as is the case in the ventricle when auricular contractions are forced. The two cycles are most nearly compensatory when the forced contraction comes late.

Of compensatory pauses.

When the forced ventricular beat comes so late in diastole that its retrograde impulse finds the node refractory, there may be compensation in the true sense of the term (Fig. 11). This phenomenon has been witnessed by Hering and Rothberger and Winterberg. We find some over-compensation by a few thousandths of a second. The rate of *A-V* nodal discharge is affected although the forced contraction wave from the ventricle does not reach this node. A disturbance, similar in some respects, was referred to under the effects of forced auricular contractions, but the direction of the change is different. With forced ventricular beats the nodal discharge slows, with forced auricular beats it quickens; such at all events is our experience of compensatory beats. A curious example of compensation is seen in Fig. 13. If this figure is compared with Fig. 14 from the same animal, a conspicuous difference will be noted in the complexes of the forced ventricular beats. Yet stimulation was applied at the same ventricular point in each instance. In Fig. 13 the ventricular complex is transitional between the usual complex and that following stimulation of the infundibulum in this animal (Fig. 14). The forced beat is premature by but a few thousandths of a second. A contraction starts at the point of stimulation, but already an impulse is well on its way to the ventricle from the *A-V* node; in the original curve the beginning of the usual auricular complex can be seen distinctly, immediately before the abrupt upstroke of the ventricular electrocardiogram. The two ventricular waves, the one following normal, the other abnormal, channels, meet in the ventricular walls.

TABLE VIII.

(COMPENSATORY BEATS). *A-V* RHYTHM. STIMULATION OF RIGHT VENTRICLE.

Dog.		Initial Cycles.		Forced Cycle.	Returning Cycle.	Restored Cycles.			
EC	<i>P-P</i>	.4645	.4647	.4640	.4710	.4654	.4674	.4648	
(1749)	<i>P-R</i>	+ .0399	+ .0400	+ .0393	— .0215	+ .0401	+ .0382	+ .0357	+ .0387
	<i>R-R</i>	.4646	.4640	.4032	.5326	.4635	.4649	.4678	
EE.	<i>P-P</i>	.6468	.6485	.6487	.6508	.6485	.6491		
(1805)	<i>P-R</i>	.0568	.0574	.0592	.0083	.0571	.0568	.0570	
(Fig. 11)	<i>R-R</i>	.6474	.6503	.5978	.6996	.6482	.6493		
The same, but with the two excitation waves meeting in the ventricle.									
EZ.	<i>P-P</i>	.5636	.5670	.5635	.5668	.5679	.5639	.5657	.5694
(2179)	<i>P-R</i>	.0307	.0313	.0298	.0309	.0159	.0299	.0313	.0314
(Fig. 13)	<i>R-R</i>	.5642	.5655	.5646	.5518	.5819	.5653	.5658	.5655
FB.	<i>P-P</i>	.4098	.4055	.4101	.4077	.4098	.4057	.4093	.4076
(2213)	<i>P-R</i>	+ .0313	+ .0324	+ .0350	— .0048	+ .0313	+ .0308	+ .0339	+ .0304
	<i>R-R</i>	.4109	.4081	.3703	.4438	.4093	.4088	.4058	.4091

DISCUSSION AND FURTHER OBSERVATIONS.

We have seen that the length of the returning cycle, whatever its origin, is mainly controlled by the rate of impulse formation in the dominant centre. We have also seen that when *A-V* rhythm is disturbed, the cycle is usually slightly longer than an initial cycle. In this connection it is a matter of indifference whether the forced contraction comes from auricle or ventricle. It has been stated too, that this customary lengthening cannot be due to alterations in the conduction of impulses from node to auricle, or node to ventricle as the case may be, for such changes would tend to produce shortening and not lengthening of the returning cycle. In the hope of ascertaining the cause of the usual lengthening, we have studied the effects of rhythmic stimulation of auricle or ventricle upon the returning and restored cycles in the same animals.

Effects of rhythmic stimulation. We find that the after effects of rhythmic stimulation to be most inconstant from animal to animal, and to be extremely complex in one and the same animal. The lengths of the first few restored cycles are *usually reduced* by several hundredths of a second; that is to say the re-awakened *A-V* rhythm is faster than before the disturbance. This fact is well illustrated in Table IX. Moreover it is the rule to find the rate *decreasing* as the rhythm becomes restored (Fig. 16, 18 and 19), until the previously established rate is resumed (see Table IV). In this respect *A-V* rhythm contrasts with the so-called idioventricular rhythm, which at its development is slow and increasing in rate as it develops. In a few experiments we have seen no alteration of rate as a result of the disturbance. In two exceptional instances (Fig. 17) we have seen a slower rhythm of development with a subsequent increase of rate, but in one of these the ventricle was exhibiting alternation. In other experiments, while the actual

rate at development was high, there was at first gradual quickening and eventually slowing. The duration and rate of the rhythmic stimulation appears to have no decided influence upon the events.

The returning cycle is as a rule increased in length, as compared to initial cycles; rhythmic stimulation is usually followed in these animals by an *A-V* rhythm whose rate is enhanced but which is slowing. The returning cycle after a solitary excitation may be reduced in length, but these are not necessarily the experiments in which rhythmic stimulation accelerates the dominant rhythm. That the altered rate at which *A-V* impulses are elaborated, subsequent to rhythmic stimulation, influences the length of the cycle returning after such rhythmic stimulation would seem natural. Nevertheless we have not been able to show that such influence is material; on the contrary our evidence is against its being so. Studying the lengths of the returning cycles following rhythmic stimulation, we find that they are usually reduced in length as compared to the returning cycle which follows a single forced beat; a good example is seen in Fig. 15 where two forced beats are followed by a shorter returning cycle than is a single beat. But we have also seen instances where the returning cycle after rhythmic stimulation was longer than that following a single forced beat, and yet the restored cycles after rhythmic stimulation were at an enhanced rate. The effects of rhythmic stimulation fail to explain a lengthened returning cycle in vagotomised animals.

In one experiment, a rhythm of apparently high nodal origin was disturbed by rhythmic ventricular beats, excited as usual from the conus. These ventricular beats failed to affect the dominant rhythm (Fig. 20 and Table XI), and the auricle continued to beat in response to the dominant impulses. There was, for the time being, complete dissociation. Yet the rate of these dominant impulses rose during the stimulation and in some instances continued to rise subsequent to the termination of stimulation; eventually they fell back to the previous rate. The influence of rhythmic stimulation may be an indirect one as this experiment shows, and is not necessarily the result of contraction waves reaching the dominant focus. These effects are comparable to the slight disturbance of accurate compensation where a forced auricular beat fails to reach the ventricle or where a forced ventricular beat fails to reach the auricle.

In many experiments rhythmic stimulation has resulted in alterations in the rate at which other centres elaborate their impulses; notably the sino-auricular node. A succession of rhythmic contractions forced from auricle or ventricle may lead at its termination to the temporary dominance of the sino-auricular rhythm (see Fig. 18 and Tables) by increasing the rate of impulse formation at the natural pacemaker. The first few beats of the restored rhythm are from the sino-auricular node, beating, considering its continued cooled condition, at an unnaturally great rate; this rhythm slows, and eventually the *A-V* node resumes its sway but also at an enhanced rate, which subsequently slows. We have seen precisely similar changes in the

TABLE IX.
A-V RHYTHM. SINGLE AND SUCCESSIVE STIMULATION OF RIGHT APPENDIX.

	Initial Cycles.			Additional Forced Cycles.		Returning Cycle.	Restored Cycle.
				Forced Cycle.			
EE. (181a) (Fig. 15)	P-P	.6666	.6656	.4291		.7746	.6624
	P-R	.0556	.0575	.0578		.1264	.0579
	R-R	.6685	.6659	.4977		.7061	.6634
EE. (181b) (Fig. 15)	P-P	.6624	.6599	.4401	.4440	.7638	.6541
	P-R	.0579	.0589	.0607	.1258	.1304	.0593
	R-R	.6634	.6617	.5052	.4486	.6927	.6560
EJ. (1971)	P-P	.6806	.6779	.3405	.4076	.7290*	.6583*
					(7 cycles)		.6738*
	P-R	.0620	.0622	.0616	.1562	.1559	.1208
	R-R	.6808	.6773	.4351	.4080	.6939	.6695
					(7 cycles)		.6655
EX. (2161b)	P-P	.4130	.4139	.3034		.4934	.4137
	P-R	.0390	.0379	.0356		.1224	.0411
	R-R	.4119	.4116	.3902		.4121	.4133
EX. (2169)	P-P	.3588	.3548	.2872		.4319	.3557
	P-R	.0640	.0647	.0648		.1196	.0468
	R-R	.3588	.3555	.3420		.3591	.3641
EX. (2166)	P-P	.4198	.4209	.3993	.3004	.5081	.4222
	P-R	.0405	.0398	.0401	.0619	.1264	.1430
	R-R	.4191	.4212	.4211	.3657	.4247	.4192
EX. (2161a)	P-P	.4181	.4195	.3984	.3024	.4973	.4156
	P-R	.0392	.0404	.0396	.0612	.1231	.0436
	R-R	.4193	.4187	.4200	.3647	.4178	.4147
EX. (2160)	P-P	.4130	.4109	.2755	.2928	.4963	.4076
	P-R	.0409	.0409	.0412	.1264	.1323	.0440
	R-R	.4130	.4112	.3607	.2948	.4080	.4039
EZ. (2183c)	P-P	.4133	.4131	.2605		.5092	.4213
	P-R	.0270	.0278	.0274		.0983	.0299
	R-R	.4141	.4127	.3314		.4408	.4198
					(3 cycles)		.4168
							.0284
							.4127
							.4156
							.4133

EZ.	P-P	.4267	.4287	.2798	.7403 (3 cycles)	.5314	.4351	.4273	.4271
(2183a)	P-R	.0288	.0288	.0286	.0974	.0993	.0317	.0290	.0282
	R-R	.4267	.4285	.3486	.7422 (3 cycles)	.4638	.4324	.4265	.4280
EZ.	P-P	.4219	.4232	.2412	.9992 (4 cycles)	.5309	.4305	.4228	
(2183b)	P-R	.0282	.0293	.0293	.1009	.1065	.0314	.0284	.0280
	R-R	.4230	.4232	.3128	.10048 (4 cycles)	.4558	.4275	.4224	
EZ.	P-P	.4356	.4330	.3197		.5240	.4419	.4362	.4366
(2184c)	P-R	.0288	.0287	.0288		.0981	.0303	.0279	.0293
	R-R	.4355	.4331	.3890		.4562	.4395	.4376	.4358
EZ.	P-P	.4306	.4296	.3883	.2511	.5370	.4429	.4305	.4293
(2184d)	P-R	.0297	.0297	.0300	.0687	.0985	.0334	.0315	.0302
	R-R	.4306	.4299	.4270	.2833	.2472	.4719	.4410	.4292
FA.	P-P	.5896	.5837	.4407		.6325	.5697	.5688	.5640
(2202a)	P-R	.0884	.0871	.0885		.0986	.0885	.0890	.0886
	R-R	.5883	.5851	.4508		.6224	.5702	.5684	.5636
FA.	P-P	.5935	.5894	.4098		.6387	.5834	.5847	.5876
(2204a)	P-R	.0866	.0860	.0866		.0968	.0852	.0842	.0864
	R-R	.5919	.5931	.4200		.6271	.5824	.5869	.5876
FA.	P-P	.5480	.5482	.5462	.5265	.5358	.5033	.5270	.5518
(2202b)	P-R	.0884	.0873	.0879	.0937 (5 cycles)	.0946	.0844	.0857	.0870
	R-R	.5469	.5488	.5471	.5314	.5256	.5046	.5283	.5501
FA.	P-P	.6356	.6282	.5800	.14076 (5 cycles)	.6345	.5971	.6126	.6246
(2204b)	P-R	.0873	.0875	.0876	.0927 (5 cycles)	.0964	.0799	.0860	.0872
	R-R	.6373	.6358	.6283	.5851 (5 cycles)	.6180	.6032	.6138	.6258
FB.	P-P	.4310	.4328	.3301		.5099	.4296	.4333	.4327
(2208b)	P-R	.0315	.0326	.0322		.1140	.0362	.0328	.0315
	R-R	.4321	.4324	.4119		.4321	.4262	.4339	.4308
FB.	P-P	.4299	.4258	.2924	.2309	.5264	.4399	.4299	.4288
(2208a)	P-R	.0308	.0304	.0313	.1221	.1355	.0438	.0370	.0335
	R-R	.4295	.4267	.3832	.2443	.4347	.4331	.4264	.4273
FB.	P-P	.4279	.4285	.3794	.3590	.5036	.4317	.4284	.4275
(2206b)	P-R	.0372	.0368	.0363	.0855 (2 cycles)	.1109	.0459	.0390	.0380
	R-R	.4275	.4280	.4286	.3853	.4386	.4248	.4274	.4271

* The auricular contractions terminating these cycles were of S-A nodal origin

TABLE X.
A-V RHYTHM. STIMULATION OF RIGHT VENTRICLE (SINGLE AND SUCCESSIVE).

Initial Cycles.		Forced Cycle.	Additional Forced Cycles.	Returning Restored Cycle.		
FA. (2196)	P-P	.5462	.5496	.5693	.5217	.5437
	P-R	+	+.0859	+.0856	—	.1160
	R-R	.5463	.5492	.5677	.7242	.5432
FA. (2194)	P-P	.5968	.6000	.5870	.6317	.5842
	P-R	+	+.0857	+.0865	—	.1021
	R-R	.5976	.6000	.4059	.3637	1.0898
FB. (2211b)	P-P	.4216	.4183	.3401	.4583	.4235
	P-R	+.0323	+.0332	+.0325	—	.0802
	R-R	.4225	.4176	.2274	.5708	.4242
FB. (2210a)	P-P	.4216	.4206	.3798	.4349	.4239
	P-R	+.0324	+.0313	+.0305	—	.0799
	R-R	.4205	.4198	.2694	.5472	.4249
FB. (2214b)	P-P	.4539	.4547	.3960	.4853	.4567
	P-R	+.0308	+.0313	+.0304	—	.0807
	R-R	.4544	.4538	.2849	.5963	.4580
FB. (2210c)	P-P	.4228	.4190	.4201	.4490	.4238
	P-R	+.0314	+.0298	+.0304	—	.0774
	R-R	.4212	.4196	.3897	.3386	1.3746

TABLE XI.
DOG FC. A-V RHYTHM. SUCCESSIVE STIMULATION OF RIGHT VENTRICLE.

Curve.	Initial Cycles.		Restored Cycles.	
(2234)	P-P	.5116	.5124	.5074
(Fig. 20)	P-R	.1102	.1104	.1090
	R-R	.5118	.5110	.5084
(2235)	P-P	.6628	.6636	.6566
	P-R	.0902	.0912	.0900
	R-R	.6638	.6624	.6566

Tachycardia,
right ventricle;
rate 188; dura-
tion 15 seconds.

Tachycardia,
right ventricle;
rate 186; dura-
tion 15 seconds.

.5015 .5043 .5081 .5047 .5061 .5357 .5366 .5383 .5407 .5417 .5449
.1115 .1125 .1122 .1110 .1099 .1092 .1110 .1113 .1108 .1099
.3185 .3207 .5758 .5091 .5044 .5049 .5346 .5359 .5401 .5410 .5412 .5440

.6749 .6470 .6269 .6097 .6082 .6098 .6090 .6138 .6190
.1089 .0928 .0899 .0909 .0928 .0880 .0929 .0899 .0923
.3243 .3218 .4301 .6309 .6240 .6107 .6101 .6050 .6139 .6108 .6214

case of centres which we are unable accurately to locate (Fig. 19) and similar changes although of a still more temporary kind may occur after single forced beats, as we have noticed. We ascribe these escapes to the same indirect quickening of the escaping centre which we have witnessed in the case of *A-V* rhythm.

Whether the reaction described is a sympathetic reflex or whether it is due to bettered nutrition as a result of raised heart rate, we have not attempted to determine. Sufficient, that the effects of rhythmic stimulation fails to help us in explaining the length of the returning cycle.

We may turn our attention to the lengths of the restored cycles following single forced contractions. When *S-A* rhythm prevails, these restored cycles are almost always longer than initial cycles, but as we have said there are exceptions. When *A-V* rhythm prevails they are usually lengthened, but frequently there may be shortening. Neither is there any constant relation between reduction or increase of the returning cycle and reduction or increase in the length of the restored cycle. We are forced to the conclusion therefore that the lengthening of a returning cycle has an independent cause and we apply this conclusion not only to *A-V* rhythm but to *S-A* rhythm also. In the latter case we believe, as already stated, that the position in which the forced beat arises sufficiently accounts for lengthening, in the case of *A-V* rhythm such an explanation does not hold, for we do not judge the length of the returning cycle by the contractions of the chamber from which the forced beat comes. If a forced auricular contraction disturbs an *A-V* impulse in the process of its creation, we should expect the subsequent impulse to be formed at the same rate; we judge of rate by measuring the interventricular interval. The returning cycle is found generally to be long by comparison with initial cycles, a similar lengthening may be found in the subsequent cycles; yet the causes of lengthening in the returning and restored cycles appear to be distinct.

Our suggestion of the cause of lengthening in the returning cycle when it occurs is put forward tentatively; it may be explained if we suppose that the impulse centre is not immediately upon the path through which impulses pass from auricle to ventricle or ventricle to auricle; that it lies a little way off this channel and that a little time is lost while the impulse is travelling to it, though meanwhile the same impulse is passing on to stimulate the second main heart chamber. The chief objection to this hypothesis is that it leaves no explanation of shortening of the returning cycle, when, as exceptionally is the case, this shortening occurs; unless the centre is in these instances in the direct path.

Finally, we may revert to the question of the returning *As-Vs* interval. When we deal with an *A-V* rhythm in which the *As-Vs* interval is long the returning *As-Vs* interval is reduced in length. This reduction of interval, like the lengthening of interval when the initial *As-Vs* intervals are short, occurs although the pause preceding it is increased only by a few thousandths or a few hundredths of a second. Further, we have recorded instances of

change in this interval where the preceding pause is unaltered or very slightly shortened (for example, Curves 2161*b* and 2169, Table IX). The changes in the *As-Vs* intervals which accompany forced heart beats are almost universally ascribed solely to the changing periods of rest which the conducting tissues have enjoyed. In the light of our observations we find this view insufficient and cannot accept it in its entirety. Changes in the returning and restored *As-Vs* intervals are often conspicuous at the termination of rhythmic stimulation; they are usually more pronounced than after single interruptions. The direction of change is governed, as in the case of single beats, by the lengths of the initial intervals. After a number of rhythmic interruptions, and subsequent to a returning cycle which is conspicuously shortened (Curve 2202*b*, Table IX) the returning and restored *As-Vs* intervals may show decided shortening under suitable circumstances. The disturbing beat appears in itself to exercise a definite influence upon conduction as upon other functions of the heart; the channels through which such effects are produced are unknown to us; but it seems evident that the familiar formulæ of rest and recovery incompletely explain the phenomena witnessed. In this connection we may remember the recognised and profound effects which interpolated contractions of ventricular origin exert upon the succeeding *As-Vs* interval. Curiously, in our own series interruptions of a compensatory character have left this interval practically unchanged. The influence whatever it may be is, if not capricious, at least elusive.

CONCLUSIONS.

The following conclusions apply to the heart of the dog freed from central vagal control.

1. Premature contractions arising in the auricle and disturbing an *S-A* rhythm, are accompanied by changes in the *As-Vs* interval. The forced interval is lengthened, the returning interval is shortened.
2. When similar premature beats disturb an *A-V* rhythm, the returning *As-Vs* interval is lengthened, providing that the original *As-Vs* intervals are conspicuously short.
3. The change in the length of the returning interval is due in both instances to bettered conduction, the difference in the direction of the change in *S-A* rhythm and *A-V* rhythm being due to the relative positions of the automatic centre and the tissue in which the conduction changes occur.

4. The tissues whose conduction is chiefly affected by the disturbance of a premature contraction are the *A-V* nodal tissues or tissues in their immediate neighbourhood.

5. The length of the returning cycle, following an extrasystole, is chiefly controlled by the rate of the dominant rhythm which such extrasystoles disturb. The excess of the returning over the initial cycle increases as the forced beat is more premature, when a forced auricular beat disturbs an *S-A* rhythm, but not when it disturbs an *A-V* rhythm.

6. In the case of *A-V* rhythm, the excess is usually small, minute or absent. That is to say the length of the returning cycle is very close to that of an initial cycle.

7. It is the rule that in *A-V* rhythm, as in *S-A* rhythm, the greater the prematurity of a forced auricular contraction the larger is the returning ventricular cycle. This observation throws doubt upon Wenckebach's hypothesis, that the variation in the length of the returning cycle in *S-A* rhythm, results from variations in the rate of conduction.

8. We can find no evidence that extrasystoles by altering the rate of the dominant rhythm materially influence the length of the returning cycle.

9. In *A-V* rhythm the usual after-effect of rhythmic stimulation of the heart is an enhanced but reducing rate of stimulus production. This effect contrasts with that witnessed in the case of an idio-ventricular rhythm.

10. A forced contraction, or series of such beats, may influence the rate at which dominant impulses are elaborated, although the forced contractions fail to travel over the position of the dominant centre. This effect upon rate disturbs perfect compensation of the disturbance.

11. Compensation does not occur, except as a rare result of accidental balance, unless the forced contraction wave fails to reach the dominant centre.

12. It seems to us questionable that the formulæ of rest and recovery or want of rest and recovery completely account for changes in the *As-Vs* intervals which accompany extrasystolic disturbances of an otherwise regular heart action.

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The electrocardiograms published were taken from Lead II. The curves are standardized so that 1 mm. = 10^{-4} volts. The time marker records either twenty-fifths of seconds, where the background is covered by small squares (Fig. 1) or fifths of seconds (Fig. 2). In certain of the figures the curves have been excised more generously to show the movements of A and the ventricular lever.

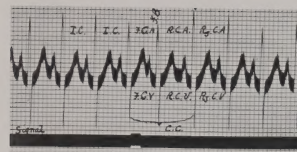


Fig. 1.

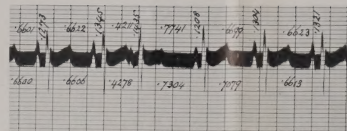


Fig. 2.

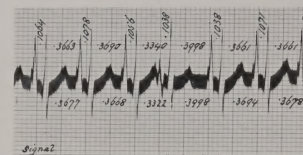


Fig. 5.

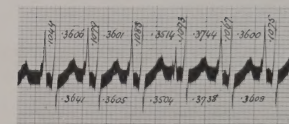


Fig. 6.

Fig. 1. (Dog EM. 2068). S-A rhythm disturbed by a single forced auricular contraction (F.B.). I.C. = initial cycle; F.C.A. = forced auricular cycle; F.C.V. = forced ventricular cycle; Re.C.A. and Re.C.V. = restored auricular and restored ventricular cycles, respectively; C.C. = conjoined cycles.

Fig. 2. (Dog EL. 1970). S-A rhythm disturbed by a forced beat of inferior caval origin. To show conspicuous shortening of the returning A-Vs interval. In this and subsequent figures the measurements are given in seconds, the auricular cycles being marked on top and horizontally, the ventricular cycles below and horizontally; the A-Vs intervals are marked vertically.

Fig. 3. (Dog EL. 2051). Simultaneous myocardiograms and electrocardiograms, showing a forced beat of inferior caval origin, disturbing A-V rhythm. The lettering above is similar to that of Fig. 1. R.Int. = returning A-Vs interval. Rs. Int. = restored A-Vs interval. The returning interval is increased in length.

Fig. 4. (Dog EL. 2044). A forced beat from the right appendix disturbing A-V rhythm. The returning A-Vs interval is increased in length.

This curve illustrates variations in the interval between the onset of P and the upstroke of the auricular lever (A), according to the position at which the auricular contraction arises. The intervals are given as P.A. in the curve.

Fig. 5, 6 and 7. (Dog EL. 2050b, 2048, and 2047). Three forced contractions disturbing S-A rhythm. The point of stimulation was in each instance the inferior cava. The shape of the natural inferior caval complex is shown in Fig. 5. In Fig. 6 and 7 there is almost exact compensation, and this has resulted because the forced contraction waves have failed to reach the pacemaker before its discharge. This fact is witnessed to by the transitional form of the electric complexes. In each case two excitation waves, one of S-A nodal and one of inferior caval origin have met in the walls of the auricle.

Fig. 8. (Dog EL. 2040). A forced auricular contraction (right appendix) interrupting A-V rhythm. The contraction wave finds the A-V node refractory.

Fig. 9. (Dog ED. 1777). A forced ventricular beat interrupting A-V rhythm and leading to temporary escape of the S-A node.

Fig. 10. (Dog EE. 1792). Two forced auricular contractions interrupting A-V rhythm. The first which is the more premature leads to quickening and escape of the S-A rhythm.

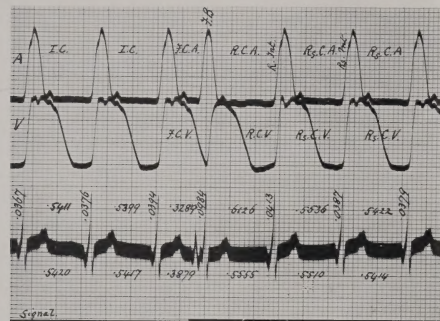


Fig. 3.

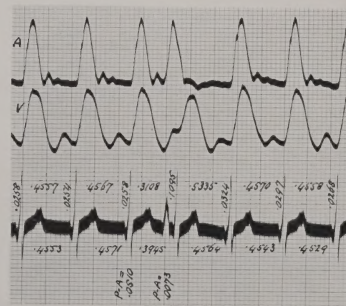


Fig. 4.

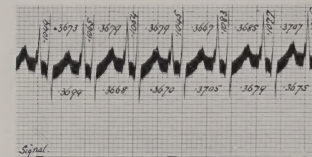


Fig. 7.

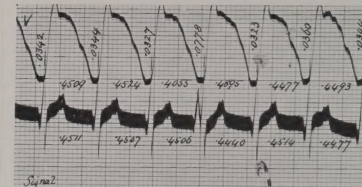


Fig. 8.

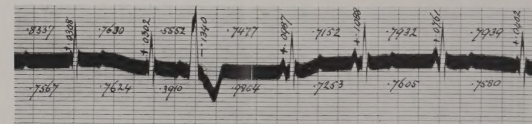


Fig. 9.

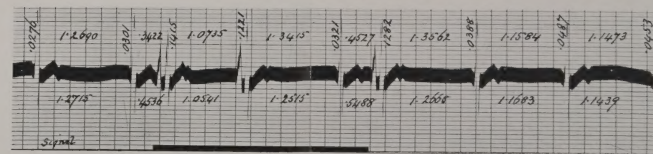


Fig. 10.



Fig. 11.

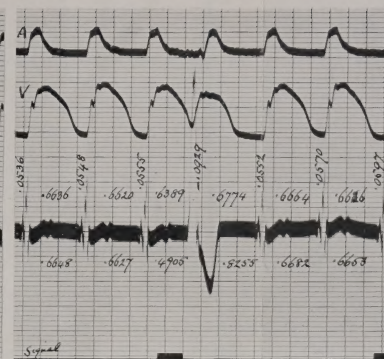


Fig. 12.

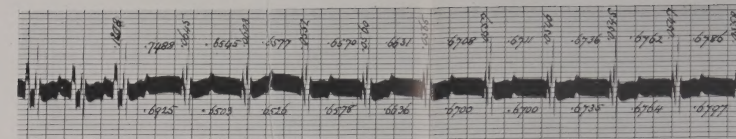


Fig. 13.

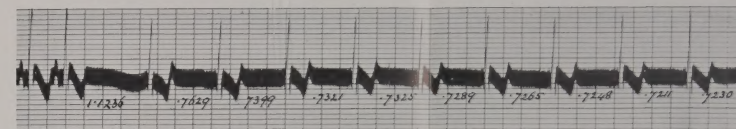


Fig. 14.

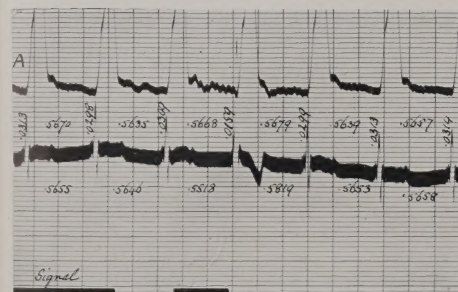


Fig. 15.

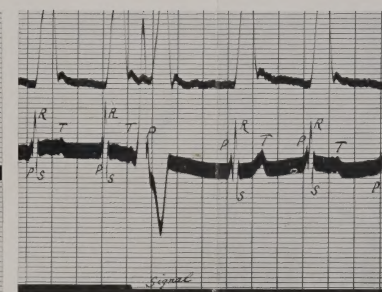


Fig. 16.

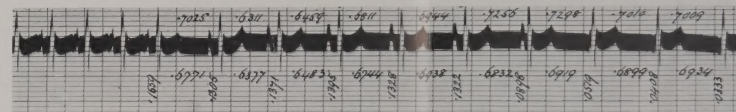


Fig. 17.

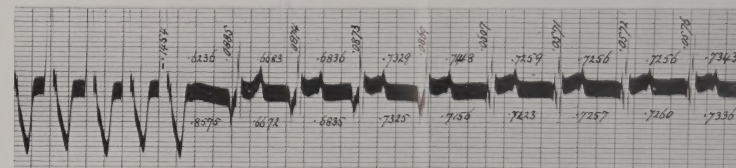


Fig. 18.

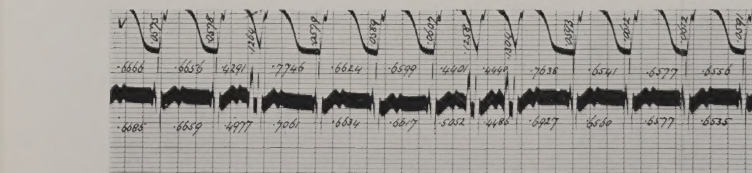


Fig. 19.

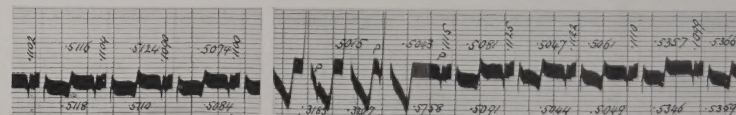


Fig. 20.

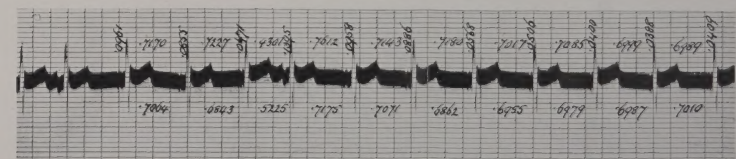


Fig. 21.

Fig. 11 and 12. (Dog EE. 1805a and 1791b). Forced ventricular beats disturbing A-V rhythm. The first disturbance (Fig. 11) is compensatory or, more strictly, a little over-compensatory.

Fig. 13 and 14. (Dog EZ. 2179 and 2180). Forced ventricular beats disturbing A-V rhythm. The first disturbance (Fig. 13) is compensatory. A comparison of the abnormal ventricular curves of Fig. 13 and 14 and of their time relations shows that in Fig. 13 two excitation waves have met in the ventricle. The transitional form of the forced complex is thus explained.

Fig. 15. (Dog EE. 1811 a and b). A single and two forced auricular contractions disturbing A-V rhythm. The single beat is followed by the longer returning pause.

Fig. 16. (Dog EE. 1801). The end of a period of forced tachycardia, started in the auricle and disturbing A-V rhythm. Showing the usual decreasing A-V rate subsequent to the disturbance.

Fig. 17. (Dog EM. 1874). An exceptional curve of similar kind showing an increasing rate subsequent to the disturbance.

Fig. 18. (Dog EJ. 1942). A similar curve showing escape of the S-A rhythm as a result of its quickening; showing also the gradual retardation of the S-A rhythm and return of the A-V rhythm.

Fig. 19. (Dog EE. 1812). The end of a period of forced tachycardia, started in the right ventricle and disturbing A-V rhythm. The restored beats are due to a quickened rhythm from a new focus. This rhythm slows and the A-V rhythm becomes re-established and subsequently slows in rate.

Fig. 20. (Dog FC. 2234). The first portion of the curve shows the original A-V rhythm. The second portion shows the end of a period of tachycardia forced from the ventricle. In this instance the original A-V impulses are undisturbed; the auricle beats over the period of tachycardia in response to the original A-V rhythm and for the moment there is dissociation. But during the accelerating of the ventricle, the auricular rate is also raised. It slows some little while after the cessation of the forced tachycardia.

Fig. 21. (Dog EJ. 1967). Forced auricular contractions occurring at frequent intervals and disturbing A-V rhythm. Each forced beat increases the returning and restored A-V intervals materially; they gradually decrease to the original length of .040 seconds.

CONTENTS

THE EFFECTS OF PREMATURE CONTRACTIONS IN VAGOTIMISED DOGS, WITH ESPECIAL REFERENCE TO ATRIO-VENTRICULAR RHYTHM	335
---	-----

BY THOMAS LEWIS AND PAUL D. WHITE.

*(Cardiographic Department, University College Hospital Medical
School.)*

OBSERVATIONS UPON VENTRICULAR HYPERTROPHY, WITH ESPECIAL REFERENCE TO PREPONDERANCE OF ONE OR OTHER CHAMBER ..	367
---	-----

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School.)*